

Aortic regurgitation and sinus of Valsalva-right atrial fistula after blunt thoracic trauma

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SUMMARY Non-penetrating chest trauma commonly causes a wide variety of cardiac injuries. Disruption of the aortic valve with resultant aortic regurgitation is not uncommon; conversely, a sinus of Valsalva-right atrial fistula, in the absence of a congenital sinus of Valsalva aneurysm, has been reported only once previously. This report describes the detection by preoperative cardiac catheterisation of both aortic regurgitation and a sinus of Valsalva-right atrial fistula after blunt chest trauma, and its surgical management. The need for preoperative cardiac catheterisation in patients suffering from non-penetrating cardiac trauma is emphasised, even when the diagnosis appears clear, because of the diverse nature and possible multiplicity of cardiac lesions.

Non-penetrating chest trauma commonly causes cardiac injury, and a wide variety of anatomical derangements may ensue.¹ The most frequent valvular lesion described in those who survive the trauma has been disruption of the aortic valve with resultant aortic regurgitation.¹ A much less common lesion resulting from blunt trauma is a tear in the aortic wall resulting in a sinus of Valsalva-right atrial fistula, with an associated left to right shunt. Only one such case has previously been reported.²

The following case report describes a patient who presented with both aortic regurgitation and a sinus of Valsalva-right atrial fistula after non-penetrating chest injury. The clinical signs of aortic valve disruption masked those of the sinus of Valsalva-right atrial fistula and the presence of the fistula was only detected by preoperative cardiac catheterisation.

Case report

A 28-year-old tall thin previously healthy white man was involved in a car accident. He sustained multiple contusions to his chest and extremities but no serious injuries were apparent. He was seen at an emergency room and discharged after his chest radiograph was reported to be normal. He felt well except for chest wall pain until two weeks after the accident. Over a three to four day period he then gradually developed progressive symptoms of pulmonary congestion which rapidly culminated in his admission to a local hospital. There he was found to be in severe pul-

monary oedema with a murmur of aortic regurgitation. After a 5 litre diuresis in 24 hours his condition improved and he was transferred to Parkland Memorial Hospital.

On arrival he was very dyspnoeic while sitting at an angle of 60 degrees. Blood pressure was 130/30/0 mmHg in both arms and pulse rate was 100 a minute, with bounding peripheral and carotid pulses. He had a high arched palate but no ectopia lentis. There was no jugular venous distension but chest examination disclosed bilateral basal râles and dullness to percussion. The apex beat was hyperdynamic in the fifth intercostal space at the midclavicular line and there was a fourth heart sound. A grade 3/6 systolic ejection murmur and a grade 3/6 long early diastolic decrescendo murmur were heard, loudest along the left sternal border, and an Austin-Flint rumble was heard at the apical area. There was no continuous murmur. Abdominal examination was normal and there was no peripheral oedema.

Chest radiographs showed the heart size to be at the upper limits of normal with bilateral pleural effusions, pulmonary venous congestion, and prominent pulmonary arteries. The electrocardiogram showed sinus tachycardia but was otherwise normal. M-mode echocardiography showed fluttering of the anterior mitral valve leaflet as well as premature closure of the mitral valve. The left atrial and aortic root dimensions were both increased at 4.2 cm, as were the left ventricular end-systolic and end-diastolic dimensions (4.3 and 6.9 cm, respectively). There was no left ventricu-

lar hypertrophy. Two dimensional echocardiography suggested a flail aortic valve leaflet. The tricuspid valve appeared normal both by M-mode and two dimensional echocardiography. A cut film aortogram was performed to rule out dissection of the aorta. This showed moderately severe aortic regurgitation but no other abnormality.

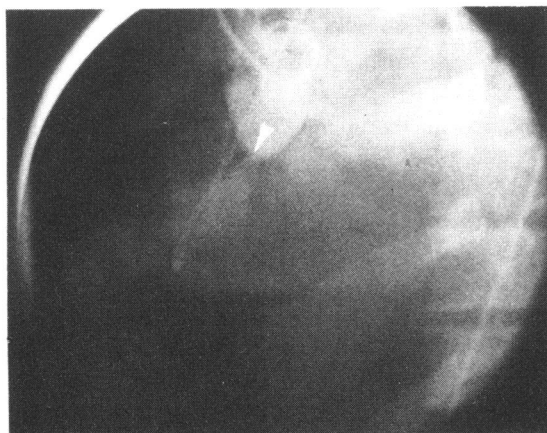
At cardiac catheterisation routine oximetry showed a significant step-up in oxygen saturation (Table). Though the saturation step-up appeared to be at the right ventricular level, the identical saturation measurements throughout the right ventricle raised the question of a more proximal shunt with streaming of blood through the upper part of the tricuspid valve. The calculated pulmonary blood flow using the Fick principle was 10 l/min, systemic blood flow was 4.5 l/min, yielding a calculated left to right shunt of 2.2:1. Indicator dilution curves suggested a 1.4:1 left to right shunt and no right to left shunt. Haemodynamic measurements showed normal right sided pressures (right atrial mean pressure 5 mmHg, pulmonary arterial pressure 27/12 mmHg); mean pulmonary capillary wedge pressure was 14 mmHg, and left ventricular end-diastolic pressure was 27 mmHg. Cineangiography of the aortic root showed slight dilatation of the aortic annulus, severe aortic regurgitation, and passage of contrast from the right sinus of Valsalva into the right atrium (Fig.). Left ventricular angiography showed no mitral regurgitation, normal wall motion, and a left ventricular ejection fraction of 0.73. The coronary arteries were angiographically normal.

At operation there was no evidence of a haemopericardium and the right sinus of Valsalva was found to bulge into the right atrium. A jet of blood from the right sinus of Valsalva could be palpated as it

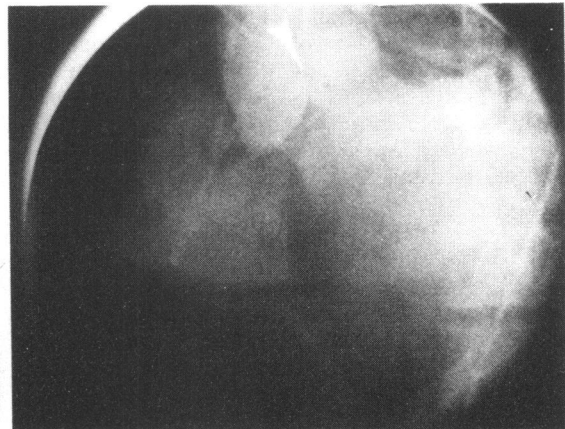
Table Oximetry measurements

	Oxygen saturation (%)
<i>First set</i>	
Left pulmonary artery	80
Main pulmonary artery	77
Right pulmonary artery	77
Right ventricular outflow tract	76
Right atrium at tricuspid valve	64
High inferior vena cava	68
Low inferior vena cava	54
<i>Second set</i>	
High superior vena cava	62
Low superior vena cava	66
Superior vena cava at right atrium	69
High right atrium	67
Mid right atrium	72
Low right atrium	69
High inferior vena cava	65
Low inferior vena cava	52
<i>Third set</i>	
Main pulmonary artery	79
Right ventricular outflow tract	78
Right ventricle mid-cavity	78
Right ventricle at tricuspid valve	77
Right ventricular apex	79
Right atrium at tricuspid valve	70
High inferior vena cava	70

entered the right atrium above the tricuspid valve. The aortic valve was bicuspid. There was a one centimetre tear in the aortic valve across the rudimentary commissure of the right cusp, with associated aortic valvular regurgitation. The aortic opening of the sinus of Valsalva-right atrial fistula was situated in the aortic wall above the right cusp. The aortic valve was excised and replaced with a 29 mm Carpentier-Edwards porcine heterograft and the fistula was closed by direct suture from the aortic side. A biopsy of the aortic wall was histologically normal. The patient had an uncomplicated postoperative course.



(A)



(B)

Fig. Supravulvar aortogram in the left anterior oblique projection. Contrast material passes from the sinus of Valsalva into the right atrium (A) and into the left ventricle via the aortic valve (B).

Discussion

Aortic regurgitation resulting from blunt chest trauma has been described both as an isolated injury and as one of a constellation of injuries.^{1,3-5} Though rupture of congenital sinus of Valsalva aneurysms is well described,^{6,7} a sinus of Valsalva-right atrial fistula resulting from penetrating trauma is comparatively rare.^{8,9} The occurrence of such a fistula after non-penetrating chest trauma is even less common: only one such case has been reported.² This was not associated with a congenital sinus of Valsalva aneurysm.

In the absence of a congenital sinus of Valsalva aneurysm which impinges on the right atrium, there is normally a space between the right atrium and the aorta. Thus, an acute tear of the proximal aorta generally leads to a leak into the pericardial space rather than into the right atrium. Therefore, the pathogenesis of a sinus of Valsalva-right atrial fistula is likely to represent a subacute process: the tear in the aortic wall results in a limited dissection of the aorta with haematoma formation, which impinges on the right atrium and subsequently ruptures into that chamber. This delayed rupture into the right atrium may have been responsible for the late deterioration in our patient's condition, which allowed full evaluation and surgical treatment, and probably explains why acute haemopericardium and cardiac tamponade did not occur.

The combination of aortic valvular regurgitation and sinus of Valsalva-right atrial fistula with a left to right shunt has not previously been described. The physical findings of the aortic regurgitation (systolic and diastolic murmurs, wide pulse pressure, and bounding pulses) masked the features of a sinus of Valsalva-right atrial fistula. Furthermore, though echocardiography suggested a torn aortic leaflet and aortic regurgitation, it did not show diastolic fluttering of the tricuspid valve (as described previously with a similar fistula²), or any protrusion of the right sinus of Valsalva. Since the severity of the patient's aortic valvular regurgitation appeared to explain his clinical status adequately, we did not suspect the presence of the fistula before cardiac catheterisation, and would not have detected the fistula before, or possibly

even at the time of operation, had catheterisation been omitted.

This case report serves to emphasise the need for preoperative cardiac catheterisation in patients suffering from non-penetrating cardiac trauma, even when the diagnosis appears clear and the patients's condition stable, because of the diverse nature and the possible multiplicity of cardiac lesions which may not be detected by non-invasive techniques.

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